A DIGEST OF RECENT WORK ON EPILEPSY.

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I propose herein to give a digest of the work done upon epilepsy in the two years from and including 1898 to the present month. In arranging a review of the literature upon epilepsy of a little more than two years, consisting of many hundreds of separate articles, I was surprised to find so little devoted to elucidating the problem of the hereditary or acquired instability of the cerebral cortex, the essential basis of most of the epilepsies. This is the more striking as it is steadily becoming more apparent to neurologists that the predisposition plays the most important rôle. It is generally conceded to-day that the hereditary element, which largely composes the predisposition, is found in 85 per cent. of all cases (Gowers).

ETIOLOGY OF EPILEPSY.

Since the discovery of the cortical motor area and the pyramidal tracts it has been generally admitted that without the cortex there can be no epilepsy, and the impulses from the cortex must travel to the peripheral nerves along the pyramidal paths; but beyond this nearly everything is still in dispute, and the investigations of different workers are so variant as to make it impossible to harmonize their conflicting views and researches.

In a study upon the motor paths in epileptic attacks, Bischoff finds that cutting the pyramidal paths at any level renders the production of muscular spasm by the faradic current more difficult; isolated muscles may be made to contract in contra-lateral extremities. Despite complete division of the paths, unilateral or bilateral, at any level, persistent faradic irritation of the cortical motor area may lead to genuine epileptic convulsions. Faradization of the pyramidal tract itself produces tonic muscular contractions in the contra-lateral extremities. He found that isolated injury to the tegmentum seems to have no influence upon the production of single spasms in the contra-lateral extremities. If the tegmentum and pyramidal paths were both divided on one side and the homo-lateral hemisphere were irritated, the convulsions were weaker on the contra-lateral side than they were on the homo-lateral side when the contra-lateral hemisphere was irritated. No convulsions were produced on irritation of the motor area when the optic thalamus, hypothalamic region and pyramidal paths of one side were destroyed.

Bilateral injury of the tegmentum and pyramid at the level of pons appeared to wholly interrupt the results of cortical irritation. Unilateral division of the tegmentum, with partial or complete injury of the homo-lateral, or of both pyramids, at any level of the crus cerebri between the thalamus and proximal end of the vagus nucleus, arrested the possibility of generating upon the operated side epileptic convulsions in the contra-lateral extremities, while direct irritation of the tegmentum led to persistent tonic contractions in contra-lateral extremities. With intact cortex and partial preservation of conducting paths to the cord isolated spasms may be produced in contra-lateral extremities by faradizing either hemisphere; but after injury (unilateral) of the tegmentum these contractions cannot be evoked. Hence we infer, with Bischoff, that the tegmentum must be a collaborator in the irritation which produces epileptic paroxysms, although from his experiments one is forced to admit that the part played by the tegmentum is relatively small.

Prus has reviewed the literature of experimental research into the functions of the corpora quadrigemina and publishes his investigations upon dogs in this connection. After careful and painstaking investigation he has educed some very valuable evidence in regard to the origin of tonic and clonic spasms, the essential features of an epileptic convulsion. Stimulation of the anterior corpora quadrigemina induced tonic spasm and true epileptic attacks. Prus applied cocaine to parts under investigation to avoid reflex action, which might tend to confuse the experiments. Stimulation of the posterior quadrigemina was followed by clonic spasm. He therefore infers, as do Bischoff and Hering, that the corpora quadrigemina contain motor centers which, under irritation in the anterior part, produce tonic cramps and in the posterior part clonic spasms. Prus experiments also elicited many other interesting facts not especially bearing upon the subject of epilepsy.

Hering, in an equally interesting series of experiments upon dogs and apes, concludes that all tracts through which muscular contractions may be brought about may also serve for muscular relaxation. Specific inhibitory paths (like the vagus inhibitory) or inhibitory centers have not as yet been proven to exist.

It should also be said that with reference to the cerebral nervous system and the musculature of the body no specific inhibitory paths have as yet been demonstrated, and the probability that such exist is small, for there would then be required a double number of nerve fibers to preside over contraction and relaxation of muscles. Inhibition can be explained without invoking the agency of special fibers for that purpose by the theory that it depends on some underlying alteration in the condition of nervous or muscular tissue; consequently, inhibition is to be explained by physiological and pathological research and not by anatomy. Neither are there demonstrable specific paths for the production of clonic spasms (cortical epilepsy). as all paths subserving movements in general can be employed in the production of clonic spasm. But as all paths do not possess the same degree of irritability, Hering logically infers that clonic spasms may be more easily produced through the pyramids than through other paths.

A very important fact has been determined by Hering's experiments, namely, that dogs and apes appear to show a difference with regard to "corticofugal" paths. In apes the pyramids appear to play a greater rôle in the induction of isolated movements of the contra-lateral extremities (whence it is to be inferred that in mankind a still greater part is played by the pyramids). On the other hand, the dog has an isolated contra-lateral path through which movements can be induced; and in the ape the contra-lateral path can functionate only in connection with the homo-lateral path. Other minor differences between the ape and dog were in evidence. The author is still prosecuting his studies with special reference to the bearing of these results upon human epilepsy. Further report will be awaited with great interest.

THE EPILEPTIC PREDISPOSITION.

Studies upon the epileptic predisposition have been made by Joffroy, in studying the relationship of alcoholism and absinthism to epilepsy. He calls attention to the marked difference between the infectious diseases like measles and those requiring a special soil for their propagation, such as tuberculosis, gout, obesity and the so-called neuroses. The underlying factor of the latter class is degeneracy.

Of the various kinds of poisoning alcohol and absinthe are most advantageous for this study. Joffroy agrees with Féré that alcoholic and absinthic epilepsy are essentially like true epilepsy at bottom—that is, due to heredity. The statistics of Martin show that 304 children (infants) of alcoholic stock 48 died of convulsions, 144 died of indifferent affections and 112 survived. Of this number the huge total of 60 cases became epileptic. Thus 108 out of the original 304 became victims of convulsive affections (*Gaz. des hôp.*, April, 1879).

Whether it is alcohol, puerperality, digestive disorders or infectious diseases, etc., it is always a poison acting upon a hereditary or acquired predisposition. Degeneracy is held to underlie neurology and psychiatry and implies inferiority, a lessening of the power of resistance. This inferiority is such that it can never be made up, and therefore leads Joffroy to say, with Esquirol, "once a degenerate always a degenerate," and epilepsy is *per se* a disease of degeneracy and therefore incurable. As a passing comment we may say that many cases do recover notwithstanding, which leads us to still study and more carefully scrutinize the peculiarities of the epileptic predisposition.

Hochaus has put forward the premature calcification of the vessels as a cause of epilepsy, and describes a case which occurred in a man of twenty-eight years. The ordinary exciting causes were absent; the man died of status epilepticus; autopsy proved the lesion in the left central convolution and cornu Ammonis. The resulting malnutrition was the cause of epilepsy and death. The point of most interest is, what caused the early calcification? Virchow has described similar cases due to osseous lesions, but his were never epileptic. Hochaus' case had no osseous lesion. The lesion described by Hochaus can hardly be commonly present in the epileptic brain, otherwise it would not have failed of previous report.

Under title of "Epilepsy of Cardiovascular Origin," Jones and Clinch have presented a study recently. The article first treats of the relationship between heart affections and epileptic attacks. This sketch also gives an account of numerous cases of slow pulse, aortic disease, etc., in association with "epileptic swoon" and other features of epilepsy. Napoleon, for example, had a normal pulse of 40 and suffered from petit mal.

This historic résumé is of much interest and value, but it is too long for extended notice here. The bearings of the subject are numerous. Thus an association of vertigo, syncope and epileptoid attacks is held to be evidence of a degenerated heart (Allbutt). Cardiac syncope certainly shades imperceptibly into petit mal.

A case illustrating the vascular apparatus at fault has been presented by G. Variat, who described a child, nineteen months old, suffering from epileptic attacks caused by congenital cyanosis. The cyanosis was due to a malformation of the heart.

In regard to the increase of intracranial pressure as a cause of epilepsy, Bayerthal has described the action of a long existant meningocele which produced epilepsy, and maintains that if such pressure is long enough continued epilepsy will invariably occur.

A more exhaustive study upon intracranial pressure has been made recently by Nawratski and Arndt, who refer to Kocher's theory, propounded in 1893, concerning the genesis of epileptic attacks; he was able to drain the lateral ventricle and the porencephalic cyst, which communicated with it, and thus cure the epilepsy. From this and other clinical facts he was led to conclude that increased tension of the cerebro-spinal fluid played an important rôle in the genesis of epilepticattacks, the other leading factor being irritability of the cerebral cortex. Kocher and others, therefore, advocated the theory of a drainage of the lateral ventricles.

Nawratski and Arndt's experiments upon three epileptics were conducted only in status epilepticus and serial epilepsy. The rise in pressure during the paroxysms was a *result* rather than a *cause*, and due to the arrested respiration and disturbance of intracranial pressure. House has recently reattempted to establish Kocher's original thesis.

Epilepsy as a symptom of cortical lesion has been thoroughly studied by Lemos, who stated that cortical lesions seated in the psychomotor zone, or without, especially the latter, may cause not only Jacksonian epilepsy but typical grand mal as well, and that the difference between partial and total epilepsy is purely theoretical.

An exceedingly interesting clinical study upon the family degeneracy that produces myoclonus or paramyoclonus and epilepsy has been most exhaustively presented by Lundborg. His paper first gives a sketch of the entire family stock that produced the fourteen cases of myoclonus and epilepsy; notes upon the seven distinct families from which the cases were recruited; a short résumé of the individual patients; lastly, general consideration of the disease, etc. The stock consisted of several generations of an intensely neuropathic family; three elaborate genealogical tables give the relationship and the neurotic peculiarities of the different individuals.

The following neuroses were especially prominent: tic convulsif, paralysis agitans, simple epilepsy and epilepsy associated with myoclonus. Other more neurotic evidences of degeneracy, such as obesity, tuberculosis, gout, etc., were present. The female sex appeared to predominate markedly over the males, the proportion being even 2 or 3 to 1. The age of nine or ten, or better, the interval of seven to fifteen was by far the most frequent period for the diseases to begin. While heredity played the main rôle (not always by direct inheritance) there were even here many excitants, such as alcoholism, syphilis, etc. There was no evidence of a paramyoclonic epileptic begetting a similarly afflicted child. But few cases were present in which a general neuropathic inheritance was entirely absent. The overwhelming influence of consanguinity in families already predisposed to diseases of degeneracy was most strikingly in evidence in Lundborg's essay. The progeny of the patients who were not childless died of miscellaneous affections (tuberculosis, tubercular meningitis, etc.); a few apparently enjoyed perfect health. In regard to the relationship of the paramyoclonus and epilepsy, some cases of epilepsy developed before myoclonus, while in other instances the myoclonus preceded the epileptic convulsions by one or two or three years, and possibly, in one or two cases, as high as eleven and twenty years. Lundborg believed that the development of myoclonus upon epilepsy stamped the individual as hopelessly degenerate and therefore incurable.

Taking this entire stock together, we find the following:

1st gen. (Per P)	healthy?	
2d gen.	healthy?	
3d gen.	healthy?	
4th	Paramyoclonus and epilepsy	6
	Tic convulsif	2
	Paralysis agitans	2
5th	Paramyoclonus and epilepsy	4
	Plain epilepsy	1
	Tic convulsif	9
	Paralysis agitans	3
6th	Paramyoclonus and epilepsy	4
	Plain epilepsy	5
	Tic convulsif	0
	Paralysis agitans	0
		36

Numerous other neurotic peculiarities are incidentally mentioned: idiocy, imbecility, paralytic dementia, suicide, etc.

While the epilepsy and paramyoclonus were undoubtedly closely associated, it could not be determined whether either one stood in any direct causal connection with the other, which is similar to the conclusion arrived at by Garnier and Santenoise in the case reported by them, yet it is practically established in Lundborg's study that epilepsy is particularly liable of occurrence in the great majority of degenerate families.

In connection with paramyoclonic epilepsy, the observations of Bechterew are of interest. He describes chorea-like convulsions in an epileptic. The association was not a coincidence. The choreic twitchings became more intense as the time for a paroxysm drew near, and after the epileptic attack had passed by the choreic movements ceased for a considerable period. The epileptic attack itself seemed to be an exacerbation of the ordinary choreic movements plus loss of consciousness; the chorea, of course, could not be held to stand in any immediate causation to the epilepsy.

TOXICS AND AUTO-TOXICS IN THE PRODUCTION OF EPILEPSY

Under this head we find most of the present day studies of the immediate excitants to epileptic convulsions. As to the definite poisons, we find a thesis by Bucelli, who states that alcoholics often become epileptics and *vice versa.* He also puts forward an alcoholic equivalent for epilepsy in those patients having a sudden impulsion of dipsomania. This is not to be confounded with true dipsomania, as the act is more automatic and the desire perfectly irresistible. Of 226 alcoholic epileptics studied by Bucelli, only five were due solely to alcohol, that is, the attacks in five appeared after indulgence in alcohol in perfectly sound persons. Ballet believes that alcohol is less frequently a potent agent in the causation of epilepsy than absinth and cocaine. He nevertheless quotes many observations that epileptics leaving asylums have attacks almost immediately, due to indulgence in alcohol.

Bourneville and Dardel and Dide have recently reported many cases in which typhoid has excited the occurrence of epileptic attacks; it is believed that the fever stops the brain development and the retrograde process gives origin to epilepsy. The rôle of typhoid in this connection must always be decidedly secondary to scarlet fever as a toxic agent causing epilepsy. An unsigned article in the *Allg. Wiener med. Zeitung,* after carefully surveying the whole field of present day research in epilepsy, puts forward gas poisoning as a cause of genuine epilepsy. An illustrative case is reported in full; all the pathological findings in similar cases on record from the same cause were reviewed, and a softening of the corpus striatum and inner capsule was found in all. Experiments showed that vascular alterations similar to lesions found in these autopsies could be produced; it was, therefore, believed by this author that permanent damage of the finer vessels of the brain might result by gas poisoning. This "chemical epilepsy" may not be dissimilar to the toxic epilepsy by alcohol which has already been exhaustively and carefully studied.

In the study of the morbid anatomical peculiarities and the toxics generated by them as a cause of epilepsy, the work of Ohlmacher is in evidence. He has published a number of articles upon the subject. Careful microscopical and macroscopical study have been and are still being made by him upon all epileptics dying in the Ohio Hospital for Epileptics. He has found eight out of eighteen cases in which there was a persistent thymus; a surprisingly large number, and altogether too large to be explained by coincidence. In the eight cases subjected to autopsy there were found present, in addition to enlarged and persistent thymus, a pronounced enlargement of the intestinal and splenic lymph follicles; more or less hypertrophy of the lymphatic glands and lymphadenoid follicles of the tongue, larynx, trachea, esophagus, and even stomach. The arteries were narrowed, fat was abundantly present and there were osseous changes indicative of old rickets. Every case did not exhibit these peculiarities, the thymus lesion being the only constant one present. These patients were below middle life, and at least six of the eight had idiopathic grand mal. Four had periodic mania; three cases were found dead in bed, having been previously in good health. The subjects of thymic asthma and thymic sudden death in children are well known. The enlarged and persistent thymus is also a part of the "status lymphaticus," or lymphatic constitution. Ohlmacher holds that the same lesions were found in the two states, at least in the eight cases upon which autopsies were made; in addition, peculiarities in the arteries and bones were also found. It remains to be seen whether or not this status lymphaticus is one of the factors at the bottom of epilepsy.

Ferrarina refers to the various examples of neuroses of hepatic origin, and adds a case of apparent hepatic epilepsy. The boy was first epileptic at the age of eight years; the type was classic with later equivalents of ambulatory automatism. The patient was first seen at the age of seventeen. He was somewhat jaundiced; between fits he had urobilinuria, and after a fit biliary pigments appeared. The entire digestive tract was in bad condition. He was placed upon purgatives and milk regimen. The seizures and hepato-intestinal symptoms improved at the same time; urobilin and bile pigment left the urine. Axenfeld (1895) has shown that bile may excite the cerebral irritability when injected into the carotid; and Bickel (1897) has corroborated this. Unfortunately no mention is made of the hereditary factors in this case, a point always essential to determine the relative severity of the toxic agency and its true rôle in the production of the epilepsy.

In the multiple studies upon the autoxic element in epilepsy, Futcher, of Johns Hopkins, attacks Rachford's paraxanthin theory in the *St. Paul's Med. Journal*, Sept., 1899. He first gives the chemical nature and relationship of the leucomains, of which group xanthin and paraxanthin are members, and states that paraxanthin acts as a tetanizer to some of the lower animals. Rachford's original paper on leucomain poisoning appeared in 1895; he attributes to the agency of paraxanthin a migraine, an epilepsy, and a gastric neurosis. Rachford quotes largely from Salomon, whose results he is able to confirm. He also thinks that Haig's "uric acid epilepsy" is of the same nature as his own paraxanthin epilepsy; but he holds that uric acid and the urates are not poisonous.

Rachford's views have recently been re-affirmed by their author, hence his older views need not be repeated in this connection. Futcher next proceeds to adduce evidence against the correctness of this leucomain theory. In 1889 Denis and Chouppe found that the toxicity of the urine was normal in epileptics. In 1890 Féré found that the preparoxysmal urine was hypertoxic, and much more toxic than the post-paroxysmal urine. Voisin and Peron found that the paroxysmal urine was hypotoxic; during the paroxysm the toxicity increased while the postparoxysmal urine was hypertoxic. Marret and Bose in 1896 found that the urine of epileptics is in general hypotoxic, and this lowered toxicity is most marked after the paroxysms and between seizures. The urine of the pre-paroxysmal period is relatively hypertoxic, and the normal toxic coefficient is lowered immediately after the fit, the toxicity being reached some hours after the paroxysm has occurred.

In attempting to harmonize these discrepant views, Futcher states that the consensus of opinion appears to be that the urine of epileptics is hypertoxic about the time of the attacks, its toxicity being much greater than during the inter-paroxysmal period. Thus far these studies of urinary toxicity appear to favor the autotoxic theory of epilepsy.[1]

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[1] The toxicity of the urine will be mentioned again under examinations of secretions in epileptics.

During the present year Pfaff and Putnam attempted to disprove Rachford's claim that paraxanthin is the cause of migraine, incidentally touching upon epilepsy in this connection. They claim that paraxanthin is found in an equal degree in the healthy and unhealthy, the inference being that Rachford's mode of analysis is fallacious.

Deutsch, in an article on auto-intoxications, describes a peculiar case in which epileptiform convulsions were present in the midst of acetonemic coma. Deutsch's patient presented the picture of genuine status epilepticus. As far as could be determined, the patient was free from any congenital or acquired taint. The author chooses to call the affection acute aceton poisoning, and borrows the expression "acetonic epilepsy" from v. Jaksch (1888). He thinks it possible that studies of the source and nature of acetonemia might throw some light on the subject of genuine epilepsy. In this connection Deutsch mentions a case of epilepsy which appeared to be due originally to sudden fright (fall in the water); yet an autopsy two years later revealed a cyst in the brain. He quotes Kranisky's theory advanced in 1897, that epilepsy was due to the periodical formation of carbonate of ammonia.

Beside Deutsch, von Jaksch, Wayner and others have described cases of acetonic epilepsy.

Weber in certain cases believes also that ammonia carbonate produced from urea causes epilepsy. Bond in many cases in which paraxanthin or xanthin urine would be excluded found leucomain poisoning. Errors of diet appeared to play the rôle of genesis of attacks. Bond states that it is precisely the idiopathic cases of epilepsy in which gastro-intestinal irritation occurs most frequently, and it is equally certain that proper treatment directed towards overcoming these factors shows us the best results.

In regard to the vasomotor origin of epilepsy which Gowers has stated is "alike unnecessary and unproven," not much has been done recently. The present status of the matter has been very well set forth by Binswanger in his monograph upon epilepsy, appearing in 1899, which gives a historical sketch of the attempt to identify vasomotor phenomena with epileptic attacks. The literature of the subject extends back to the time of Astley Cooper.

After a careful reading of recent literature upon the subject, we would conclude that the problem of the vasomotor relationship and arterial pressure upon the production of epilepsy is still to be solved. It appears in all the numerous observations and experiments that the cortical discharge takes effect upon the peripheral vascular territories rather than upon cortical or vasomotor medullary centers. In fairness to the believers of the vasomotor theory it may be said that there is a small number of cases of epilepsy which may be ascribed to vasomotor disturbance, especially in those cases where the entire vascular system is involved. This is not only true of grand mal but also of petit mal and psychic epilepsy. In those cases there is turgescence of the skin, profuse sweating and dicrotic pulse. In such cases a sharp rise of temperature is usually found.

We conclude with Binswanger and Bechterew that the only type of epileptic convulsion in which the medullary vascular center is primarily involved is in the reflex species which are certainly exceedingly rare.

MANIFESTATIONS OF EPILEPSY.

In reviewing the different manifestations of epileptic phenomena, we find the psychical equivalents have received a great deal of attention; of late, several voluminous works have appeared. Bombarda has published a small volume containing a study on the sleep states in epileptics, consisting of nightmare, starting in sleep, pavor nocturnus or night-terrors, seminal emissions and erotic dreams without ejaculation of semen. These he holds are nothing more than equivalents of petit mal epilepsy. He therefore believes a nocturnal pollution to be a true attack which is quite in accord with the views of Zuccurelli and Hamilton on the subject, but which is generally, and it appears to us justly discredited by conservative neurologists.

Many cases of retarded epilepsy have been reported of late, principally studied by Guillon and Allen, but the studies upon them have not especially enriched our present day knowledge of the essential nature of epilepsy. Accompanying the vascular changes generally found there is almost a marked predisposition.

Toulouse and Marchand and Péon have recently reported cases of general paralysis in youths who were also epileptic; but as juvenile paresis is almost always luetic (at least it is so in Mott's opinion, by whom the subject has been recently thoroughly and exhaustively studied), it seems doubtful if the cases were other than juvenile general paretics in which epileptiform convulsions were very prominent.

Féré has held for some time that laughter may be an equivalent in many epileptics and has recently reported one unique case of this kind of epilepsy. Equivalents as well as autoxics in epileptics appear to be the *bête noire* of many present day investigators in epilepsy.

Lemoine has reported the gastric forms of epileptic paroxysms; in most instances such cases appear to be the aborted gastric or epigastric aura of true epilepsy, and not deserving of special classification.

Bandropadhia reports a possible case of tetanoid epilepsy first described by Prichard. The case was apparently cured by application of iron rings on the toes where the fit began with a distinct aura. The fact of the cure in the case alone makes one look askance at the diagnosis of true epilepsy.

Von Bechterew, in a careful study of epileptic and epileptoid paroxysms of morbid fear, concludes that such are not infrequent in epilepsy; they may exist as an aura or alternate as equivalents with comitial paroxysms. In rare instances minor epilepsy may exist as paroxysmal fear alone. As a rule, loss of consciousness or vertigo do not occur. This form of epilepsy, as has been determined by many writers, is refractory to the usual anti-epileptic treatment. Bechterew insists on the essential difference between this form of epileptic fear and that accompanying neurasthenia. Féré also deals quite exhaustively with the subject, and details cases somewhat similar to those of Bechterew.

Féré describes and discusses at considerable length three cases of narcolepsy or paroxysmal sleep as equivalents of idiopathic epilepsy. The cases are rare, unique and quite without rational explanation as yet.

Higier describes a case of periodic paralysis not consecutive to convulsions. He refers to the experiments of Sherrington (1893) and Mislawski (1898), to the effect that if a given portion of the cerebral cortex is electrically excited, some muscles act spasmodically while others are relaxed; if the flexors contract, the opposing extensors are relaxed. He therefore concludes that if the intensity of the cortical irritant is of low degree paralysis may result instead of convulsion. This may in fact be demonstrated in part by experiment.

Cestan and Le Sourd discuss at great length the clinical value of the "toe phenomenon" of Babinski, and give the result, among other diseases of the nervous system, of examination of a number of cases of epilepsy (the toe phenomenon is an extension of the toes instead of flexion upon pricking the sole of the foot). Babinski maintained that there was a close clinical connection between the toe phenomenon and "spinal epilepsy," mainly because the toe phenomenon showed as the first symptoms of pyramidal involvement in disease of the nervous system. Cestan and Le Sourd, therefore, hold with Babinski that in partial epilepsies dependent upon organic changes in the pyramids the toe phenomenon is of great importance in diagnosis. This is especially valuable when we remember that even now many organic lesions upon which the so-called idiopathic epilepsies really depend go unrecognized because of the great difficulties which surround their detection.

Hughlings-Jackson has reported and described "uncinate epilepsy" cases in which there is slight asphyxia, subjective odor, chewing movements and flow of saliva, and an association of the so-called "dreamy state" in this type. Autopsy on one typical case proved the lesion true to the diagnosis before death (Jackson and Coleman).

J. W. McConnell also describes several cases of transient paralysis as epileptic equivalents, the cause of these epilepsies he attributes to auto-intoxication.

Several articles have recently appeared upon the type of epilepsy accompanied by, or consisting of, apparently purposeful movements of locomotion, such as running forward, backward, sidewise; spinning and rolling before convulsions or while in the fit, or even without a fit. Wittner in an article upon chronic diffuse post-epileptic encephalitis relates a case of procursive epilepsy in which, contrary to the title, he proves it to be secondary to organic mischief as above noted in the title. Bourneville has long believed that procursive epilepsy gradually produces encephalitis.

Ziehen in a thesis which contains his well known hypothesis based on experiments that clonic spasms are cortical, and tonic spasms of infracortical source, holds that procursive movements are infracortical in origin. In a more recent article of last year, Schuster and Mendel reported four cases of procursive epilepsy. Contrary to usual experiments, bromides were of signal use in their treatment. These authors, without the privilege of autopsy upon any of their cases, but with apparent logic, believe that this phenomenon of epilepsy is but one phase of true epilepsy.

A bizarre and rare form of epilepsy, long known, although but little commented upon of late, is the so-called attacks of hunger or states of hunger in varied associations with epileptic phenomenon. Hunger-states seem closely allied to attacks of fear described by Bechterew and others. The hunger-state should not be confounded with boulimia, a singular craving for food, common in epileptics and other neurotic people, nor with pica, a craving for unnatural food, seen in some pregnant women. Féré has reported several cases of the "hungry evil" in which it existed both as aura and as an equivalent. In this latter phase it alternated with classic attacks of epilepsy.

Two studies have been made recently in regard to the analogy of epilepsy and eclampsia; one by Dide in pointing out their similarities of heredity and auto-toxic bases, cites a case in which eclampsia gave origin to epilepsy; cases illustrating the above point have long since been placed on record. The infrequency of such an association leads one to look for other factors favoring the relationship. Under the exceptional circumstances of eclampsia causing epilepsy, Chambrelent, in reviewing the whole subject of pregnancy and epilepsy, found that some fifty epileptic women had been carefully observed while pregnant. In over half of the number the epileptic condition was benefited. Having decided on the whole, pregnancy exerts a favorable influence upon the epilepsy, Chambrelent finds that the epilepsy has no effect upon pregnancy. Even a status case did not abort. A great number of children born of epileptics have convulsions and some die from them. Probably in time statistics will be forthcoming in which some relationship may be found to exist between cases which became worse from pregnancy, and those who bear children who in turn develop convulsions. The subject is not only one of considerable scientific interest, but also of much practical importance.

It has long been a study to ascertain why epileptics have their attacks at night only. In such cases many theories have been advanced to explain the phenomenon which have been detailed at length by Pick, but none has met with general favor. It is generally conceded from all statistics that the hours 9 P. M. and 5 A. M. are the ones in which patients most frequently have attacks. Neither circulatory disturbances (Féré), nor depth of sleep (Kohlschutter, Czerney, Howell) seem to be sufficient to cover the explanation necessary. In fact, in regard to the latter theory, the frequency of attacks seems to be in inverse ratio to the depth of sleep.

Some work far from harmonious has been done upon the toxicities of the blood, urine and sweat, in recent years. Lui claims to have demonstrated marked variations in the alkalinity of the blood which seemed to have provoked acute psychoses and epileptic paroxysms. Weber maintains after careful observations that ammonia carbonate produced from urea, is the toxic agent which causes many epilepsies. Mairet and Vires infer from test of the blood serum of epileptics that epileptic blood loses some of its normal toxicities and in accord with the results of Herter's experiments they find the urine of epileptics is hypotoxic.

Voisin, in his study of the elimination of methylene blue in epileptics, injected during the attacks and the intervals between, finds a coincidence of his experiments with those of Bar in eclampsia; namely, that the elimination of methylene agrees with the lowered toxicity of the urine after an attack, showing defective elimination of poisonous substances, the evidence of an auto-intoxication.

In studying the relationship of migraine to that of epilepsy, Rachford thinks that paraxanthin ought to be found after paroxysms of epilepsy; but many observers of late have found it is infrequently the case, paraxanthin epilepsy being much more infrequent than paraxanthin migraine.

Mavrojannis concludes after a most painstaking series of observations that the sweat of epileptics has but feeble toxic power, much less than Cabitto found. Injections of sweat in rabbits produced extension of the vertebral column only, not convulsions; but even extension of the vertebral column was not obtained with the sweat of normal subjects.

Uniformity in the presence or absence of post-epileptic albuminuria is wanting; authorities are about equally divided as to its presence and absence after paroxysms. It is generally held with Pio Galante that the albumin of the post-paroxysmal state, when found, is due more to the respiratory arrest than to the increased vascular tension in the fit. Many theories and many supporters of each theory still hold prominent place. The great majority of neurologists still deny its habitual presence in the post-paroxysmal state of all epileptics.

As to post-paroxysmal exhaustion in epilepsy, it is manifested in many ways and of late has been well studied. Exhaustion-paralysis is shown in the skeletal muscles, ocular exhaustion, aural exhaustion, exhaustion of cutaneous sensibility and the rare phenomenon post-epileptic dysesthesia, recently studied by Féré. Post-epileptic pain and sensitiveness is a rarer phenomenon than anesthesia, which in turn, is of quite uncommon occurrence. The motor phenomenon of post-paroxysmal exhaustion is the most frequent of exhaustion phenomena. All the exhausted states may occur isolated or in varied combinations. They are subjects which deserve very close study and attention, as they are not infrequently indices to the brain lesion causing epilepsy.

Féré with others, has recently urged all the different forms of post-epileptic exhaustion phenomena as equivalents as well as sequella of paroxysms. There is still much to be done to solve the problem of relationships of equivalents and epileptic crises.

Rennie has recently published an interesting paper on hypotonia in epileptics. For many years hypotonicity as a post-epileptic sequel indicating the exhaustion has been written upon by continental physicians under the head of post-epileptic rigidity, and contractures as paralytic equivalents. Rennie reports three cases highly illustrative of the title of his paper.

(To be continued)

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58 RECHERCHES THERMO-ÉLECTRIQUES SUR LE CERVEAU D'UN EPILEPTIQUE. Dr. G. Mirto (Arch. italiennes de biologie, Tome XXXII, Fasc. II, 26 Fevrier, 1900; pp. 335-340).

G. Lombardini, an epileptic, aged fifteen years, has a gap in his skull (caused by a fall in early infancy), irregular in shape, nine centimeters by four, and situated, with its long axis antero-posterior, in the parietal region. Above and behind it is over a small part of the parietal ascending convolution; behind it involves almost the whole of the superior parietal convolution and the highest portion of the inferior convolution. Convulsive attacks occur at intervals of days or of weeks, while attacks of epileptic vertigo are more common. He is mentally feeble and his vision is very defective.

Dr. Mirto studied the thermal changes in the cortex under various conditions. He employed a thermo-electric pile of forty couples of bismuth and antimony plates connected with a mirror-galvanometer of such sensitivity that a rise of one degree in temperature caused a movement of about 200 mm. on the scale. This was about the largest deviation he had ever obtained from cerebral changes, the instrument being applied to the hair-covered scalp.

The observation here recorded was made while the author was watching the scale, and consisted of a deflection of 42 mm. in the positive direction, corresponding to a rise of temperature of 0.2° on the surface of the scalp. It reached its maximum in 50 seconds, and in 115 seconds decreased to 8 mm. During this time the subject had undergone an epileptic seizure without convulsions; he was pale, with closed eyes, and in a state of semi-stupor, and had passed his urine.

Thus is confirmed by accident under ideal conditions the conclusions of Mosso arrived at from experiments on dogs, that an epileptic seizure is accompanied by a cortical rise of temperature independently of muscular innervation, for in this case no convulsions occurred. Dr. Mirto concludes as a corollary that the heat-producing process of the cortex is or may be independent, both of motor discharge and of psychic concomitance, as Mosso had already maintained.

G. V. N. DEARBORN.

59 SUR L'IRRITABILITÉ. Note by Prof. A. Stefani (Arch. italiennes de biologie, XXXII, III, 26 Fevrier, 1900, pp. 439-450).

In this "note" Professir Stefani reviews the theory of irritability elaborated by Hering and published in 1889, basing the phenomena of irritability on automatic changes in the nutrition of the tissue, and practically claims that as early as 1879 he himself had published a similar doctrine on this subject. G. V. N. DEARBORN.